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| NEWS | 1 | | Web Page URLs for STN Seminar Schedule - N. America |
| NEWS | 2 | | "Ask CAS" for self-help around the clock |
| NEWS | 3 | SEP 09 | CA/CAPLUS records now contain indexing from 1907 to the present |
| NEWS | 4 | DEC 08 | INPADOC: Legal Status data reloaded |
| NEWS | 5 | SEP 29 | DISSABS now available on STN |
| NEWS | 6 | OCT 10 | PCTFULL: Two new display fields added |
| NEWS | 7 | OCT 21 | BIOSIS file reloaded and enhanced |
| NEWS | 8 | OCT 28 | BIOSIS file segment of TOXCENTER reloaded and enhanced |
| NEWS | 9 | NOV 24 | MSDS-CCOHS file reloaded |
| NEWS | 10 | DEC 08 | CABA reloaded with left truncation |
| NEWS | 11 | DEC 08 | IMS file names changed |
| NEWS | 12 | DEC 09 | Experimental property data collected by CAS now available in REGISTRY |
| NEWS | 13 | DEC 09 | STN Entry Date available for display in REGISTRY and CA/CAPLUS |
| NEWS | 14 | DEC 17 | DGENE: Two new display fields added |
| NEWS | 15 | DEC 18 | BIOTECHNO no longer updated |
| NEWS | 16 | DEC 19 | CROPU no longer updated; subscriber discount no longer available |
| NEWS | 17 | DEC 22 | Additional INPI reactions and pre-1907 documents added to CAS databases |
| NEWS | 18 | DEC 22 | IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields |
| NEWS | 19 | DEC 22 | ABI-INFORM now available on STN |
| NEWS | 20 | JAN 27 | Source of Registration (SR) information in REGISTRY updated and searchable |
| NEWS | 21 | JAN 27 | A new search aid, the Company Name Thesaurus, available in CA/CAPLUS |
| NEWS | 22 | FEB 05 | German (DE) application and patent publication number format changes |
| NEWS EXPRESS | | | DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003 |
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FILE 'USPATFULL' ENTERED AT 09:34:13 ON 17 FEB 2004

CA INDEXING COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

=> s IL-10 (p) (antibod?)

L1 5599 IL-10 (P) (ANTIBOD?)

=> s l1 (p) (IL-2)

L2 2256 L1 (P) (IL-2)

=> s l1 (p) (IL-2 antibod?)

L3 15 L1 (P) (IL-2 ANTIBOD?)

=> d l3 1-15 bib ab

L3 ANSWER 1 OF 15 MEDLINE on STN

AN 2000114114 MEDLINE

DN 20114114 PubMed ID: 10649854

TI [Collagen in the treatment of rheumatic diseases--oral tolerance].
Kolagen v liecbe reumatickych chorob--oralna tolerancia.

AU Stancikova M; Stancik R; Gubzova Z; Rovensky J

CS Research Institute of Rheumatic Diseases, Piestany, Slovakia..
stancikova@vurch.sk

SO BRATISLAVSKE LEKARSKE LISTY, (1999) 100 (10) 567-71.

Journal code: 0065324. ISSN: 0006-9248.

CY Slovakia

DT Journal; Article; (JOURNAL ARTICLE)

LA Slovak

FS Priority Journals

EM 200002

ED Entered STN: 20000218

Last Updated on STN: 20000218

Entered Medline: 20000210

AB The term "oral tolerance" means antigen specific suppression of immune response after oral application of antigen. Primary mechanisms by which oral tolerance is mediated include: deletion, anergy and active cellular suppression. The determining factor in this process is the dose of applied antigen. High doses of antigen develop deletion and anergy of cells while low doses of antigen result in bystander suppression. Recently bystander suppression has attracted attention in the treatment of autoimmune diseases. This process is connected with induction of regulatory T cells of Th2/Th3 phenotypes in gut with characteristic profile of anti-inflammatory cytokines as IL-4, IL-10 and TGF-beta. By means of circulation the lymphocytes enter the affected place and when meeting again with the antigen, they produce the same profile of cytokines which they originally made in the gut. These cytokines then suppress local autoimmune and inflammatory reaction independently of the antigen type. After successful trials of treatment with low doses of orally applied collagen type II in animal models of

experimental arthritis, this treatment was also studied in clinical trials in humans with rheumatoid arthritis. Although the results obtained to this date are very promising they can not be considered final. Several questions still need to be solved: identification of responders, determination of character and amount of collagen applied as well as the route of application. Another promising therapeutic approach could be the simultaneous application of collagen and the compounds enhancing the cell response of Th2 or Th3 lymphocytes such as TGF-beta, **IL-2, antibodies** to IL-12 which can augment the oral tolerance. In clinical praxis the treatment of osteoarthritis with collagen type I has also been successfully applied. Induction of oral tolerance is new approach in the treatment of rheumatoid arthritis and as each new therapy, it requires refinement. In the future it is expected that an improved study design and a better understanding of the underlying mechanisms of oral tolerance will lead to an increased efficacy of the therapy in humans similar to the effectiveness previously demonstrated in animal models.

L3 ANSWER 2 OF 15 MEDLINE on STN
 AN 1999262266 MEDLINE
 DN 99262266 PubMed ID: 10330268
 TI IL-2 may be a limiting factor precluding lymphocytes from genetically resistant mice from responding to HgCl2.
 AU Jiang Y; Moller G
 CS Department of Immunology, Wenner-Gren Institute, Arrhenius Laboratories for Natural Sciences, Stockholm University, 106 91 Stockholm, Sweden.
 SO INTERNATIONAL IMMUNOLOGY, (1999 May) 11 (5) 627-33.
 Journal code: 8916182. ISSN: 0953-8178.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199906
 ED Entered STN: 19990712
 Last Updated on STN: 19990712
 Entered Medline: 19990623
 AB It is unclear how HgCl2 causes autoimmune disorders in genetically predisposed rodents. We investigated the cytokine profile induced by HgCl2 in vitro, and found a high frequency of IL-2-secreting cells in splenocytes from susceptible A.SW and BALB/c mice, whereas the frequency was low in cells from resistant DBA/2 mice. More IL-2-secreting cells were induced in splenocytes from the high responder A.SW mice than in cells from the intermediate responder BALB/c mice. Unexpectedly, a similar level of IL-4 production was induced in splenocytes from BALB/c and DBA/2 mice. IL-4 production was high in unstimulated cells from A.SW mice and was further increased by HgCl2. IFN-gamma-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-gamma and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures enhanced the proliferative response to HgCl2 in splenocytes from DBA/2 mice to a level comparable with that in cells from BALB/c mice. We found no evidence for the suggestion that HgCl2 induces a Th1/Th2 imbalance in resistant/susceptible strains. We conclude that IL-2 may be a limiting factor precluding lymphocytes from resistant mice from responding to HgCl2.

L3 ANSWER 3 OF 15 MEDLINE on STN
 AN 94001805 MEDLINE

DN 94001805 PubMed ID: 8104472
 TI Brucella abortus induces a novel cytokine gene expression pattern characterized by elevated IL-10 and IFN-gamma in CD4+ T cells.
 AU Svetic A; Jian Y C; Lu P; Finkelman F D; Gause W C
 CS Department of Microbiology, Uniformed Services University of the Health Sciences, Bethesda, MD 20814.
 NC AI21328 (NIAID)
 SO INTERNATIONAL IMMUNOLOGY, (1993 Aug) 5 (8) 877-83.
 Journal code: 8916182. ISSN: 0953-8178.
 CY ENGLAND: United Kingdom
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199310
 ED Entered STN: 19940117
 Last Updated on STN: 19950206
 Entered Medline: 19931028
 AB Immunization of BALB/c mice with killed Brucella abortus (BA) has previously been shown to increase serum IgG2a levels and long-term T cell clones from these mice secrete Th1-associated cytokines: IFN-gamma and IL-2 but not IL-4 or IL-5. We analyzed cytokine gene expression following primary immunization with BA to determine when CD4+ T cells first express cytokine genes and whether specific hypothesized cytokine patterns (e.g. Th precursor, Th0) could be identified prior to a Th1-like pattern. Our results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-gamma in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-gamma or **IL-10** as early as 4 days after immunization. These results suggest that a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and that **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 4 OF 15 MEDLINE on STN
 AN 93294307 MEDLINE
 DN 93294307 PubMed ID: 8099937
 TI Regulation of IL-5 in onchocerciasis. A critical role for IL-2.
 AU Steel C; Nutman T B
 CS Laboratory of Parasitic Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892.
 SO JOURNAL OF IMMUNOLOGY, (1993 Jun 15) 150 (12) 5511-8.
 Journal code: 2985117R. ISSN: 0022-1767.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Abridged Index Medicus Journals; Priority Journals
 EM 199307
 ED Entered STN: 19930806
 Last Updated on STN: 19950206
 Entered Medline: 19930720
 AB The cytokine profiles of PBMC obtained from individuals "immune" to Onchocerca volvulus infection were compared to those from infected individuals. The immune individuals had significantly higher levels of both IL-2 and IL-5 in response to parasite Ag than did those individuals with active infection (mean IL-2 = 1.3 and 0.138 U/ml, respectively; mean IL-5 = 973 and 147.4 pg/ml, respectively), and there was a direct correlation between the production of IL-2 and IL-5. To examine the mechanism underlying the possible association between these two cytokines

in patients infected with onchocerciasis, reverse transcription followed by polymerase chain reaction was used to measure IL-5 mRNA. In response to rIL-2, IL-5 mRNA appeared as early as early as 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 was unable to induce mRNA expression for IL-4, IFN-gamma, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis, IL-5 mRNA expression was measured in PBMC stimulated with parasite Ag. Up-regulation of IL-5 mRNA was seen in all patients (peaking at 72 h) in response to Ag stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production is required to induce IL-5 and further implicates IL-5 as a possible mediator of protection in onchocerciasis.

L3 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
 AN 1999:367654 CAPLUS
 DN 131:143361

TI IL-2 may be a limiting factor precluding lymphocytes from genetically resistant mice from responding to HgCl2
 AU Jiang, Yun; Moller, Goran
 CS Department of Immunology, Wenner-Gren Institute, Arrhenius Laboratories for Natural Sciences, Stockholm University, Stockholm, 106 91, Swed.
 SO International Immunology (1999), 11(5), 627-633
 CODEN: INIMEN; ISSN: 0953-8178

PB Oxford University Press
 DT Journal
 LA English

AB It is unclear how HgCl2 causes autoimmune disorders in genetically predisposed rodents. We investigated the cytokine profile induced by HgCl2 in vitro, and found a high frequency of IL-2-secreting cells in splenocytes from susceptible A.SW and BALB/c mice, whereas the frequency was low in cells from resistant DBA/2 mice. More IL-2-secreting cells were induced in splenocytes from the high responder A.SW mice than in cells from the intermediate responder BALB/c mice. Unexpectedly, a similar level of IL-4 production was induced in splenocytes from BALB/c and DBA/2 mice. IL-4 production was high in unstimulated cells from A.SW mice and was further increased by HgCl2. IFN-γ-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-γ and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures enhanced the proliferative response to HgCl2 in splenocytes from DBA/2 mice to a level comparable with that in cells from BALB/c mice. We found no evidence for the suggestion that HgCl2 induces a Th1/Th2 imbalance in resistant/susceptible strains. We conclude that IL-2 may be a limiting factor precluding lymphocytes from resistant mice from responding to HgCl2.

RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN
 AN 1993:647694 CAPLUS
 DN 119:247694

TI Brucella abortus induces a novel cytokine gene expression pattern characterized by elevated IL-10 and IFN-γ in CD4+ T cells

AU Svetic, Antonela; Jian, Y. C.; Lu, P.; Finkelman, F. D.; Gause, W. C.
CS Dep. Microbiol. Med., Univ. Health Sci., Bethesda, MD, 20814, USA
SO International Immunology (1993), 5(8), 877-83
CODEN: INIMEN; ISSN: 0953-8178
DT Journal
LA English
AB Immunization of BALB/c mice with killed B. abortus (BA) has previously been shown to increase serum IgG2a levels, and long-term T cell clones from these mice secrete Th1-associated cytokines: IFN- γ and IL-2 but not IL-4 or IL-5. The authors analyzed cytokine gene expression following primary immunization with BA to determine when CD4+ T cells first express cytokine genes and whether specific hypothesized cytokine patterns (e.g. Th precursor, Th0) could be identified prior to a Th1-like pattern. The authors' results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated IL-10 and IFN- γ in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-IL-2 antibodies had no effect on the cytokine gene expression pattern, although treatment with anti-IFN antibodies resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN- γ or IL-10 as early as 4 days after immunization. Thus, a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and IL-10 is specifically elevated in certain Th1-like responses.

L3 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1993:537210 CAPLUS

DN 119:137210

TI Regulation of IL-5 in onchocerciasis. A critical role for IL-2

AU Steel, Cathy; Nutman, Thomas B.

CS Lab. Parasit. Dis., Natl. Inst. Allergy Infect. Dis., Bethesda, MD, 20892, USA

SO Journal of Immunology (1993), 150(12), 5511-18

CODEN: JOIMA3; ISSN: 0022-1767

DT Journal

LA English

AB The cytokine profiles of PBMC obtained from individuals immune to Onchocerca volvulus infection were compared to those from infected individuals. The immune individuals had higher levels of both IL-2 and IL-5 in response to parasite antigens (Ag) than did those individuals with active infection (mean IL-2 = 1.3 and 0.138 U/mL, resp.; mean IL-5 = 973 and 147.4 pg/mL, resp.), and there was a direct correlation between the production of IL-2 and IL-5. To examine the mechanism underlying the possible association between these 2 cytokines in patients with onchocerciasis, reverse transcription followed by polymerase chain reaction was used to measure IL-5 mRNA. In response to rIL-2, IL-5 mRNA appeared as early as 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing antibodies to IL-2. IL-2 could not induce mRNA expression for IL-4, IFN- γ , IL-10, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis, IL-5 mRNA expression was measured in PBMC stimulated with parasite Ag. Up-regulation of IL-5 mRNA was seen in all patients (peaking at 72 h) in response to Ag stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-IL-2 antibodies. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production is required to induce IL-5 and further implicate IL-5 as a possible mediator of protection in onchocerciasis.

L3 ANSWER 8 OF 15 USPATFULL on STN

AN 2004:1816 USPATFULL
 TI Prevention or treatment of cancer using integrin alphavbeta3 antagonists
 in combination with other agents
 IN Woessner, Richard, Lafayette, CO, UNITED STATES
 Kiener, Peter, Doylestown, PA, UNITED STATES
 Dormitzer, Melissa, Germantown, MD, UNITED STATES
 Walsh, William, Sharpsburg, MD, UNITED STATES
 Heinrichs, Jon, North Potomac, MD, UNITED STATES
 PA MedImmune, Inc. (U.S. corporation)
 PI US 2004001835 A1 20040101
 AI US 2003-379189 A1 20030304 (10)
 PRAI US 2002-361859P 20020304 (60)
 US 2002-370398P 20020405 (60)
 US 2003-444265P 20030130 (60)
 DT Utility
 FS APPLICATION
 LREP PENNIE AND EDMONDS, 1155 AVENUE OF THE AMERICAS, NEW YORK, NY, 100362711
 CLMN Number of Claims: 44
 ECL Exemplary Claim: 1
 DRWN 8 Drawing Page(s)
 LN.CNT 6588

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to methods and compositions designed for the treatment, management or prevention of cancer. The methods of the invention comprise the administration of an effective amount of one or more antagonists of Integrin α .sub.V β .sub.3 alone or in combination with the administration of an effective amount of one or more other agents useful for cancer therapy. The invention also provides pharmaceutical compositions comprising one or more antagonists of Integrin α .sub.V β .sub.3 and/or one or more other agents useful for cancer therapy. In particular, the invention is directed to methods of treatment and prevention of cancer by the administration of a therapeutically or prophylactically effective amount of one or more antagonists of Integrin α .sub.V β .sub.3 alone or in combination with standard and experimental therapies for treatment or prevention of cancer. Also included are methods for screening for epitope-specific Integrin α .sub.V β .sub.3 antagonists which can be used according to the methods of the invention. In addition, methods for facilitating the use of Integrin α .sub.V β .sub.3 antagonists in the analysis of Integrin α .sub.V β .sub.3 expression in biopsies of animal model and clinical study samples are also contemplated.

L3 ANSWER 9 OF 15 USPATFULL on STN
 AN 2003:237907 USPATFULL
 TI Compositions and methods for the therapy and diagnosis of colon cancer
 IN King, Gordon E., Shoreline, WA, UNITED STATES
 Meagher, Madeleine Joy, Seattle, WA, UNITED STATES
 Xu, Jiangchun, Bellevue, WA, UNITED STATES
 Secrist, Heather, Seattle, WA, UNITED STATES
 Jiang, Yuqiu, Kent, WA, UNITED STATES
 PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)
 PI US 2003166064 A1 20030904
 AI US 2002-99926 A1 20020314 (10)
 RLI Continuation-in-part of Ser. No. US 2001-33528, filed on 26 Dec 2001,
 PENDING Continuation-in-part of Ser. No. US 2001-920300, filed on 31 Jul
 2001, PENDING
 PRAI US 2001-302051P 20010629 (60)
 US 2001-279763P 20010328 (60)
 US 2000-223283P 20000803 (60)
 DT Utility
 FS APPLICATION
 LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300,
 SEATTLE, WA, 98104-7092

CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 8531

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

L3 ANSWER 10 OF 15 USPATFULL on STN

AN 2003:219754 USPATFULL

TI Tissues or organs for use in xenotransplantation

IN Liljedahl, Monika, La Jolla, CA, UNITED STATES

Marcantonio, Daniela, San Diego, CA, UNITED STATES

Aspland, Simon Eric, San Diego, CA, UNITED STATES

PI US 2003153044 A1 20030814

AI US 2002-303686 A1 20021121 (10)

RLI Continuation-in-part of Ser. No. US 2002-147286, filed on 14 May 2002, PENDING

PRAI US 2001-291394P 20010514 (60)

US 2001-312125P 20010813 (60)

US 2002-367090P 20020321 (60)

DT Utility

FS APPLICATION

LREP KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614

CLMN Number of Claims: 73

ECL Exemplary Claim: 1

DRWN 14 Drawing Page(s)

LN.CNT 5751

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides cells, tissues or organs for use in cell therapy or xenotransplantation in which at least one gene comprising an antigenic determinant recognized by a recipient organism has been disrupted. The present invention also includes methods of administering such cells and transplanting such tissues or organs in which genes encoding antigenic determinants recognized by the recipient organism have been disrupted.

L3 ANSWER 11 OF 15 USPATFULL on STN

AN 2003:134089 USPATFULL

TI Tissues or organs for use in xenotransplantation

IN Liljedahl, Monika, La Jolla, CA, UNITED STATES

Marcantonio, Daniela, San Diego, CA, UNITED STATES

Aspland, Simon Eric, San Diego, CA, UNITED STATES

PI US 2003092174 A1 20030515

AI US 2002-147286 A1 20020514 (10)

PRAI US 2001-291394P 20010514 (60)

US 2001-312125P 20010813 (60)

US 2002-367090P 20020321 (60)

DT Utility

FS APPLICATION

LREP KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614

CLMN Number of Claims: 55

ECL Exemplary Claim: 1

DRWN 14 Drawing Page(s)

LN.CNT 3786

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides cells, tissues or organs for use in cell therapy or xenotransplantation in which at least one gene comprising an antigenic determinant recognized by a recipient organism has been disrupted. The present invention also includes methods of administering such cells and transplanting such tissues or organs in which genes encoding antigenic determinants recognized by the recipient organism have been disrupted.

L3 ANSWER 12 OF 15 USPATFULL on STN

AN 2003:106233 USPATFULL

TI Compositions and methods for the therapy and diagnosis of pancreatic cancer

IN Benson, Darin R., Seattle, WA, UNITED STATES
 Kalos, Michael D., Seattle, WA, UNITED STATES
 Lodes, Michael J., Seattle, WA, UNITED STATES
 Persing, David H., Redmond, WA, UNITED STATES
 Hepler, William T., Seattle, WA, UNITED STATES
 Jiang, Yuqiu, Kent, WA, UNITED STATES

PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

PI US 2003073144 A1 20030417

AI US 2002-60036 A1 20020130 (10)

PRAI US 2001-333626P 20011127 (60)
 US 2001-305484P 20010712 (60)
 US 2001-265305P 20010130 (60)
 US 2001-267568P 20010209 (60)
 US 2001-313999P 20010820 (60)
 US 2001-291631P 20010516 (60)
 US 2001-287112P 20010428 (60)
 US 2001-278651P 20010321 (60)
 US 2001-265682P 20010131 (60)

DT Utility

FS APPLICATION

LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092

CLMN Number of Claims: 17

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 14253

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for the therapy and diagnosis of cancer, particularly pancreatic cancer, are disclosed. Illustrative compositions comprise one or more pancreatic tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly pancreatic cancer.

L3 ANSWER 13 OF 15 USPATFULL on STN

AN 2002:272801 USPATFULL

TI Compositions and methods for the therapy and diagnosis of colon cancer

IN Stolk, John A., Bothell, WA, UNITED STATES
 Xu, Jiangchun, Bellevue, WA, UNITED STATES
 Chenault, Ruth A., Seattle, WA, UNITED STATES
 Meagher, Madeleine Joy, Seattle, WA, UNITED STATES

PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

PI US 2002150922 A1 20021017

AI US 2001-998598 A1 20011116 (9)

PRAI US 2001-304037P 20010710 (60)
 US 2001-279670P 20010328 (60)
 US 2001-267011P 20010206 (60)
 US 2000-252222P 20001120 (60)

DT Utility

FS APPLICATION

LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300,
SEATTLE, WA, 98104-7092

CLMN Number of Claims: 17

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 9233

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for the therapy and diagnosis of cancer, particularly colon cancer, are disclosed. Illustrative compositions comprise one or more colon tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly colon cancer.

L3 ANSWER 14 OF 15 USPATFULL on STN

AN 2002:243051 USPATFULL

TI Compositions and methods for the therapy and diagnosis of ovarian cancer

IN Algate, Paul A., Issaquah, WA, UNITED STATES

Jones, Robert, Seattle, WA, UNITED STATES

Harlocker, Susan L., Seattle, WA, UNITED STATES

PA Corixa Corporation, Seattle, WA, UNITED STATES, 98104 (U.S. corporation)

PI US 2002132237 A1 20020919

AI US 2001-867701 A1 20010529 (9)

PRAI US 2000-207484P 20000526 (60)

DT Utility

FS APPLICATION

LREP SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300,
SEATTLE, WA, 98104-7092

CLMN Number of Claims: 11

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 25718

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Compositions and methods for the therapy and diagnosis of cancer, particularly ovarian cancer, are disclosed. Illustrative compositions comprise one or more ovarian tumor polypeptides, immunogenic portions thereof, polynucleotides that encode such polypeptides, antigen presenting cell that expresses such polypeptides, and T cells that are specific for cells expressing such polypeptides. The disclosed compositions are useful, for example, in the diagnosis, prevention and/or treatment of diseases, particularly ovarian cancer.

L3 ANSWER 15 OF 15 USPATFULL on STN

AN 2000:87729 USPATFULL

TI Method of converting a Th2-type allergic immune response into a Th1-type immune response

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PI US 6086898 20000711

AI US 1999-339068 19990623 (9)

PRAI US 1998-90390P 19980623 (60)

DT Utility

FS Granted

EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Ewoldt, Gerald R.

LREP Bozicevic, Field & Francis, Sherwood, Pamela

CLMN Number of Claims: 19

ECL Exemplary Claim: 1

DRWN 17 Drawing Figure(s); 10 Drawing Page(s)

LN.CNT 1767

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for the treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells. Immunotherapy with adjuvants, as provided in the present invention, greatly inhibits the development of airway hyperreactivity and airway inflammation. Such immunotherapy is shown to reverse ongoing airway disease, and convert allergic inflammatory responses into protective immune responses. Conditions of particular interest include allergic conditions associated with production of Th2 cytokines and/or IgE antibodies, asthma, allergic rhinitis, and anaphylactic reactions. The addition of adjuvant induces a Th1-type immune response and can redirect an established Th2-type response to a Th1-type response for the selected antigen. Preferably, antigen-specific IgE production is reduced without altering the intensity of the antigen-specific proliferative response. One particularly preferred adjuvant for use in accordance with the present invention is a *Listeria* adjuvant.

=> d his

(FILE 'HOME' ENTERED AT 09:34:00 ON 17 FEB 2004)

FILE 'MEDLINE, CAPLUS, USPATFULL' ENTERED AT 09:34:13 ON 17 FEB 2004

L1 5599 S IL-10 (P) (ANTIBOD?)
L2 2256 S L1 (P) (IL-2)
L3 15 S L1 (P) (IL-2 ANTIBOD?)

=> d l3 1-15 kwic

L3 ANSWER 1 OF 15 MEDLINE on STN
AB . . . connected with induction of regulatory T cells of Th2/Th3 phenotypes in gut with characteristic profile of anti-inflammatory cytokines as IL-4, **IL-10** and TGF-beta. By means of circulation the lymphocytes enter the affected place and when meeting again with the antigen, they. . . the simultaneous application of collagen and the compounds enhancing the cell response of Th2 or Th3 lymphocytes such as TGF-beta, **IL-2**, **antibodies** to IL-12 which can augment the oral tolerance. In clinical praxis the treatment of osteoarthritis with collagen type I has. . .

L3 ANSWER 2 OF 15 MEDLINE on STN
AB . . . by HgCl2. IFN-gamma-secreting cells were detectable in splenocytes from all three strains after activation by HgCl2. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl2. In contrast, **antibodies** against IL-4, IFN-gamma and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures. . .

L3 ANSWER 3 OF 15 MEDLINE on STN
AB . . . a Th1-like pattern. Our results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-gamma in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-gamma or **IL-10** as

early as 4 days after immunization. These results suggest that a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and that **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 4 OF 15 MEDLINE on STN

AB . . . 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 was unable to induce mRNA expression for IL-4, IFN-gamma, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis, . . . stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production. . .

L3 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN

AB . . . by HgCl₂. IFN-γ-secreting cells were detectable in splenocytes from all three strains after activation by HgCl₂. The highest frequency of **IL-10**-secreting cells was found in splenocytes from A.SW mice after activation, whereas the frequency was lower in cells from BALB/c mice, followed by cells from DBA/2 mice. We showed that neutralizing anti-**IL-2 antibody** profoundly inhibited the in vitro response to HgCl₂. In contrast, **antibodies** against IL-4, IFN-γ and **IL-10** did not significantly affect the response of splenocytes from either A.SW or DBA/2 mice. The addition of IL-2 into cultures. . .

L3 ANSWER 6 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN

AB . . . Th1-like pattern. The authors' results demonstrated a highly consistent and novel pattern of Th1/Th2 cytokine gene expression characterized by elevated **IL-10** and IFN-γ in CD4+ T cells which rapidly manifests itself and is sustained for at least 10 days after immunization. No elevation in IL-2 cytokine gene expression was observed and treatment of BA-immunized mice with blocking anti-**IL-2 antibodies** had no effect on the cytokine gene expression pattern, although treatment with anti-IFN **antibodies** resulted in increased IL-4, IL-5, and IL-9 cytokine gene expression, in the absence of any change in IFN-γ or **IL-10** as early as 4 days after immunization. Thus, a whole pathogen may trigger sufficient costimulatory signals to rapidly induce effector T cells in the absence of elevated IL-2 and **IL-10** is specifically elevated in certain Th1-like responses.

L3 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2004 ACS on STN

AB . . . 3 h after stimulation of patient PBMC, reaching a peak at 24 h; further, this response was inhibited with neutralizing **antibodies** to IL-2. IL-2 could not induce mRNA expression for IL-4, IFN-γ, **IL-10**, or granulocyte-macrophage-CSF. To assess whether IL-2 was specifically responsible for the up-regulation of Ag-induced IL-5 production in patients with onchocerciasis, . . . stimulation and was found to be independent of proliferation to Ag; in addition, this up-regulation was specifically inhibited by neutralizing anti-**IL-2 antibodies**. Further, the primary source of IL-5 mRNA was determined to be CD4+ T cells. These findings suggest that IL-2 production. . .

L3 ANSWER 8 OF 15 USPATFULL on STN

DETD . . . propranolol, and puromycin homologs, and cytoxan. Examples of non-chemotherapeutic immunomodulatory agents include, but are not limited to, anti-T cell receptor **antibodies** (e.g., anti-CD4 **antibodies** (e.g., cM-T412 (Boeringer), IDEC-CE9.1® (IDEC and SKB), mAB 4162W94, Orthoclone and OKTcd4a (Janssen-Cilag)), anti-CD3

antibodies (e.g., Nuvion (Product Design Labs), OKT3 (Johnson & Johnson), or Rituxan (IDEC)), anti-CD5 **antibodies** (e.g., an anti-CD5 ricin-linked immunoconjugate), anti-CD7 **antibodies** (e.g., CHH-380 (Novartis)), anti-CD8 **antibodies**, anti-CD40 ligand monoclonal **antibodies** (e.g., IDEC-131 (IDEC)), anti-CD52 **antibodies** (e.g., CAMPATH 1H (Ilex)), anti-CD2 **antibodies** (e.g., MEDI-507 (Medimmune, Inc., International Publication Nos. WO 02/098370 and WO 02/069904), anti-CD11a **antibodies** (e.g., Xanelim (Genentech)), and anti-B7 **antibodies** (e.g., IDEC-114) (IDEC)); anti-cytokine receptor **antibodies** (e.g., anti-IFN receptor **antibodies**, anti-IL-2 receptor **antibodies** (e.g., Zenapax (Protein Design Labs)), anti-IL-4 receptor **antibodies**, anti-IL-6 receptor **antibodies**, anti-IL-10 receptor **antibodies**, and anti-IL-12 receptor **antibodies**), anti-cytokine **antibodies** (e.g., anti-IFN **antibodies**, anti-TNF- α **antibodies**, anti-IL-1 β **antibodies**, anti-IL-6 **antibodies**, anti-IL-8 **antibodies** (e.g., ABX-IL-8 (Abgenix)), anti-IL-12 **antibodies** and anti-IL-23 **antibodies**)); CTLA4-immunoglobulin; LFA-3TIP (Biogen, International Publication No. WO 93/08656 and U.S. Pat. No. 6,162,432); soluble cytokine receptors (e.g., the extracellular domain . . . IL-6 receptor or a fragment thereof); cytokines or fragments thereof (e.g., interleukin (IL)-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-12, IL-15, IL-23, TNF- α , TNF- β , interferon (IFN)- α , IFN- β , IFN- γ , and GM-CSF); and anti-cytokine **antibodies** (e.g., anti-IL-2 **antibodies**, anti-IL-4 **antibodies**, anti-IL-6 **antibodies**, anti-IL-10 **antibodies**, anti-IL-12 **antibodies**, anti-IL-15 **antibodies**, anti-TNF- α **antibodies** and anti-IFN- γ **antibodies**), and **antibodies** that immunospecifically bind to tumor-associated antigens (e.g., Herceptin®). In certain embodiments, an immunomodulatory agent is an immunomodulatory agent other than. . .

L3 ANSWER 9 OF 15 USPATFULL on STN

SUMM [2042] **For example**, certain amino acids may be substituted for other amino acids in a protein structure without appreciable loss of interactive binding capacity with structures such as, for example, antigen-binding regions of antibodies or binding sites on substrate molecules. Since it is the interactive capacity and nature of a protein that defines that protein's biological functional activity, certain amino acid sequence substitutions can be made in a protein sequence, and, of course, its underlying DNA coding sequence, and nevertheless obtain a protein with like properties. It is thus contemplated that various changes may be made in the peptide sequences of the disclosed compositions, or corresponding DNA sequences which encode said peptides without appreciable loss of their biological utility or activity.

TABLE 1

Amino Acids

Codons

| | | | |
|-----------------------------|-----|---|---------------------------------------|
| Alanine | Ala | A | GCA GCC GCG GCU |
| Cysteine | Cys | C | UGC UGU |
| Aspartic acid GAU | Asp | D | GAC |
| Glutamic acid | Glu | E | GAA GAG |
| Phenylalanine | Phe | F | UUC UUU |
| Glycine | Gly | G | GGA GGC GGG GGU |
| Histidine | His | H | CAC CAU |
| Isoleucine | Ile | I | AUA AUC AUU |
| Lysine | Lys | K | AAA AAG |
| Leucine | Leu | L | UUA UUG CUA CUC CUG CUU |
| Methionine. . . CCU | | | |
| Glutamine | Gln | Q | CAA CAG |
| Arginine | Arg | R | AGA AGG CGA CGC CGG CGU |
| Serine | Ser | S | AGC AGU UCA UCC UCG UCU |
| Threonine | Thr | T | ACA ACC ACG ACU |
| Valine | Val | V | GUA GUC GUG GUU |
| Tryptophan | Trp | W | UGG |
| Tyrosine | Tyr | Y | UAC UAU |

L3 ANSWER 10 OF 15 USPATFULL on STN

DETD . . . Erythropoietin (EPO), anemic conditions; insulin: islets or the pancreas can be transplanted into diabetic patients; tumor necrosis factor α (TNF- α) **antibodies**, for example, for inflammatory diseases such as rheumatoid arthritis and Crohn's disease; **antibodies** against protein products encoded by oncogenes such as C-erbB-2, for example used for breast cancer and other cancers; anti-CD4 **antibodies** for example, for rheumatoid arthritis or psoriasis; anti-human Epidermal Growth Factor Receptor type 2 **antibodies**, for example, for breast cancer and other cancers; anti-Interleukin **antibodies**, such as anti-IL-1, anti-IL-8, anti IL-10, anti-IL-12 and anti-IL-15 to be used, for example, in inflammatory diseases, such as autoimmune diseases, rheumatoid arthritis, psoriasis, inflammatory bowel disease and in cancerous disease; anti-Interleukin 15 receptor anti-bodies for use against lymphoma and other malignancies, for example; anti-CD20 **antibodies** to be used, for example, for hemolytic anemia in autoimmune diseases and other hematopoietic disorders such as leukemia and lymphomas; anti-isotypic IGE **antibodies** for allergy; anti-LG914 **antibodies** for arteriosclerosis; Interferon- α for chronic hepatitis C, hairy cell leukemia and AIDS-related Kaposi's sarcoma and chronic myelogenous leukemia (CML), for. . . in donor animal such as pig of proteins, lipids and carbohydrates to induce tolerance in xenotransplantation; anti-CD40, CD28, CD25 and IL-2 **antibodies** and OKT3; anti-idiotypic **antibodies** against naturally formed **antibodies**; anti-isotypic IgG, IgM and IgA **antibodies**. The preceding is a

non exclusive list of some exemplary gene therapy applications.

L3 ANSWER 11 OF 15 USPATFULL on STN

DETD . . . Erythropoietin (EPO), anemic conditions; insulin: islets or the pancreas can be transplanted into diabetic patients; tumor necrosis factor α (TNF-alpha) **antibodies**, for example, for inflammatory diseases such as rheumatoid arthritis and Crohn's disease; **antibodies** against protein products encoded by oncogenes such as C-erbB-2, for example used for breast cancer and other cancers; anti-CD4 **antibodies** for example, for rheumatoid arthritis or psoriasis; anti-human Epidermal Growth Factor Receptor type 2 **antibodies**, for example, for breast cancer and other cancers; anti-Interleukin **antibodies**, such as anti-IL-1, anti-IL-8, anti IL-10, anti-IL-12 and anti-IL-15 to be used, for example, in inflammatory diseases, such as autoimmune diseases, rheumatoid arthritis, psoriasis, inflammatory bowel disease and in cancerous disease; anti-Interleukin 15 receptor anti-bodies for use against lymphoma and other malignancies, for example; anti-CD20 **antibodies** to be used, for example, for hemolytic anemia in autoimmune diseases and other hematopoietic disorders such as leukemia and lymphomas; anti-isotypic IGE **antibodies** for allergy; anti-LG914 **antibodies** for arteriosclerosis; Interferon- α for chronic hepatitis C, hairy cell leukemia and AIDS-related Kaposi's sarcoma and chronic myelogenous leukemia (CML), for. . . in donor animal such as pig of proteins, lipids and carbohydrates to induce tolerance in xenotransplantation; anti-CD40, CD28, CD25 and IL-2 **antibodies** and OKT3; anti-idiotypic **antibodies** against naturally formed **antibodies**; anti-isotypic IgG, IgM and IgA **antibodies**. The preceding is a non exclusive list of some exemplary gene therapy applications.

L3 ANSWER 12 OF 15 USPATFULL on STN

SUMM [2043] **SEQ ID NO:2003 is the determined cDNA sequence of clone 61496359**

L3 ANSWER 13 OF 15 USPATFULL on STN

SUMM [2044] **SEQ ID NO:1997 is the determined cDNA sequence for clone 62227174 R0394:B12**

L3 ANSWER 14 OF 15 USPATFULL on STN

SUMM [2043] **SEQ ID NO: 2004 represents the cDNA sequence for clone AA165409.**

L3 ANSWER 15 OF 15 USPATFULL on STN

DETD Anti-IFN- γ mAb R46A2 (HB170, ATCC), and anti-IL-4 mAb (11B11), were prepared from serum-free culture supernatants by ammonium sulfate precipitation. Monoclonal anti-IL-2 **antibody** S4B6 and anti-IFN- γ **antibody** XMGI.2 were obtained from Dr. Tim Mosmann (Univ. of Alberta, Edmonton, Canada). Anti-IL-4 mAb BVD4-1D11 and BVD6-24G2 were obtained from DNAX Research Institute, Palo Alto, Calif. Each of these **antibodies** was purified from ascites by ammonium sulfate precipitation and ion-exchange chromatography. Anti-IL-10 mAb SXC.1 (DNAX) was purified by ammonium sulfate precipitation followed by Sepharose 4B chromatography. Anti-IL-10 mAb 2A5 was purchased from Pharmingen (San Diego, Calif.). Neutralizing anti-IL-12 mAb C17.8 was purified from ascites by affinity chromatography.. . .